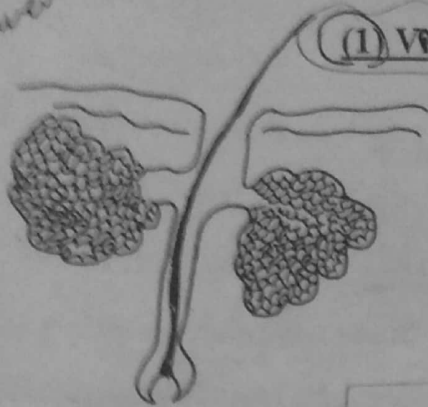


Shaving → hair + skin + hypodermis layer → after start 8 hrs

Acne and acneform eruptions

Anatomy of pilosebaceous apparatus

Types of pilo-sebaceous follicles



(1) Vellus follicle

Fine
Soft hair covering
most of the body of
children
Small
Seb gland

Sebaceous gland
is derived not developed
↳ T is not caps

At puberty

In sebaceous areas :
Face, shoulders, chest
& upper back (sites of
acne vulgaris)

Vellus hair
become

(2) Sebaceous follicle



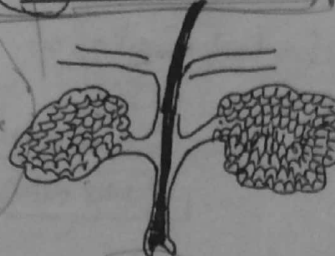
- ① Has large gland &
- ② rudimentary follicles

multi like female but
not in bearded area
& pilosebaceous
follicles

In scalp, beard,
eyebrows, axillae &
pubis

Vellus hair
become

(3) Terminal follicles



Has large gland &
powerful hair follicle.

Pilo-sebaceous
not closed
by grubby
hair

hair
Thick
long
Coarse
Tall hair

XXX X
acne

N.B. : Acne does not develop in
these follicle as the
powerful erecting hair
allows free excretion of
sebum.

X acne

Types of scar of acne :

acne

- Sebaceous secretion produced by glandular cell destruction.
- Free seb. Gland: meibomian, Tyson, female genitalia & areola.
 (Montgomery glands)
 i.e. open directly on skin surface
 ① of eyelids ② of prepuce
- Composition of sebum: Mixture of lipids (Wax, esters & cholesterol).

Function of sebum:

- ① Antifungal & antibacterial. → Sebum has fungistatic effect on staph. T. capitis. Rise after puberty
- ② Protective value. → sebums capable of metabolizing, synthesis vit D metabolites 1,25 dihydroxy vitamin D₃
- ③ Moisturizing the skin. → sebum gland → secrete vit E

Endocrinal control of sebum production:

① Androgen:

- At puberty → ↑↑ glandular size & seb. Production.
- At adult → No effect (gland was stimulated maximally by endogenous androgen).

Conversion of Testosterone → DHT
 30 Times higher in acne skin than normal skin

② Progesterone → ↑ Seb. Production.

③ Estrogen → ↓ seb. Production. by ↓ endogenous androgen production

④ Glucocorticoids → ↓ seb. Production through either direct

① effect or through suppressing adrenal androgen.

⑤ Pituitary hormones → ↑ seb. Production through either direct effect or through release of gonadal, adrenal or thyroid hormones.

⑥ Insulin like growth factors → correlate ↑ Androgen production in women

⑦ PPAR • Peroxisome proliferator activated receptor "P" → expressed on seb. gland

$$\text{Sebum production} = \frac{A+P+P}{E+G}$$

↑ Sebum Production by PPAR agonist in DM2 type

⊕ sebaceous gland & Androgen production & Keratinocyte proliferation

milk contain → estrogen
 Progesterone
 IGF-1

Acne vulgaris

*** Definition :** Inflammatory disorder of pilo-seb apparatus characterized by the formation of comedones erythematous papules & pustules, less frequently nodules or cyst.

* Epidemiology :

Adolescence in both sex.

* Aetiopathogenesis:

(1) Increased sebum production : (At puberty)

• Acne patients secrete more sebum than normal, the level of secretion correlates with the severity of acne.

• This occur due to :

① ↑ Androgen production :

- Male from testis.
- Female from suprarenal gland.

② ↑ Sensitivity of androgenic receptor of seb. Glands to the normal androgen level.

(2) Ductal hypercornification (comedone formation) :

Ductal cornification results from :

① ↑ Androgen production.

② ↑ irritant effect of sebum (especially squalene & squalene oxide),

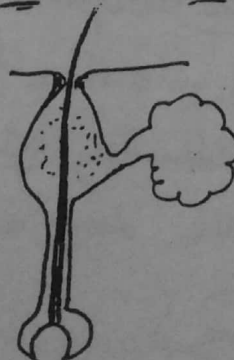
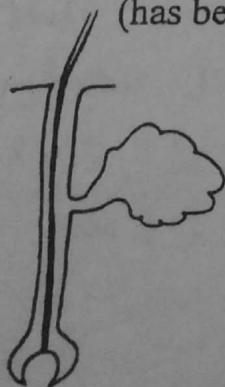
③ Deficiency in essential linolenic acid in sebum

(has been demonstrated in comedones)

→ This deficiency is reversed by isotretinoin or cyproterone acetate.

"excessive accumulation of ductal corn"

Ductal hypercornification there is increase formation & or inadequate separation of ductal corneocytes.



P. acnes binds up to the sebaceous gland (TG)

FFAs
characteristic to sebaceous gland
promote further bacterial colonization

(3) Proliferation of P. acnes

Gave, non volatile product propylgammaglutamate

Blocking of sebaceous follicles by ductal hypercornification will create an anaerobic microenvironment which will favor the proliferation of P. acnes.

Colonization of P. acnes is involved in the initiation of inflamed acne lesions.

Propionibacterium acnes

(4) Inflammation:

inflammatory events precede hyperkeratinization

Severe

Proliferated P. acnes will liberate lipase which converts triglyceride in sebum to free fatty acid → Erosion & disruption of follicular wall → escape of the follicular content → production of non specific inflammatory reaction.

ductal rupture not necessary for initiation of inflammation

Early inflammation

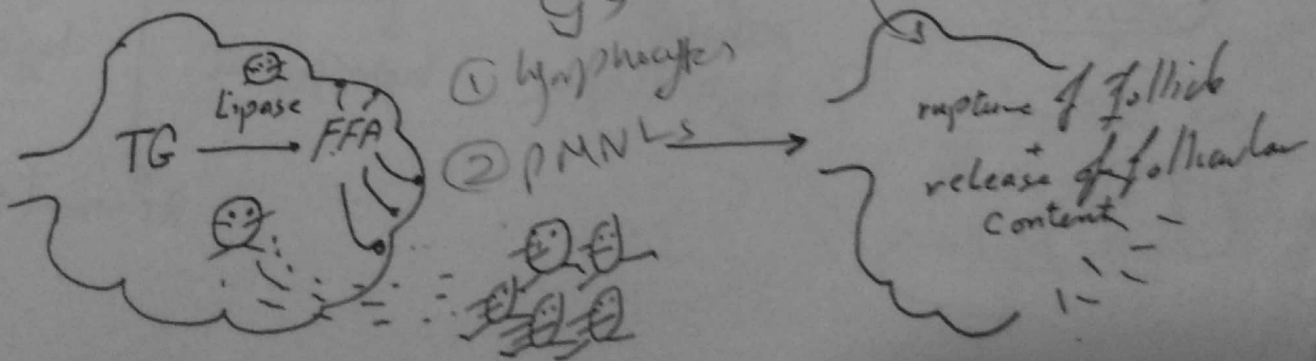
Proliferated P. acnes will also release chemotactic factors within the duct which diffuse into the dermis attracting lymphocytes & polymorphs with production of inflammation.

PMN

lymphocyte

Severe non specific inflammation

- More severe degree of inflammation result from rupture of the follicle and liberation of the follicular contents into the dermis. Early in the inflammation. Lymphocytes are involved, but within 24-48 hours the PMNLs become active.



Other etiologic factors :

- ① Genetic → rare family history
- ② Stress → ↑ acne
- ③ Sweating. Premenstrual flare → 40% of ♀
- ④ Diet

Clinical picture :

- Polymorphic rashes comedones, papules, pustules or cyst
 - Comedones consist of blackheads (open comedones) in which the black color is due to melanin not direct & white heads (closed comedones).
 - Inflammatory lesions may be superficial or deep.
 - Superficial lesions are usually papules or pustules, while deep lesions are nodules or deep pustules deep inflammatory lesions are after associated with scarring.

Grading of acne vulgaris :

Grading	Comedones	Papules/pustules	Nodules
<u>Mild</u>	Few to ≤ 25 numerous	Few to several ≤ 10	None X
<u>Moderate</u>	Numerous and/or > 25 extensive	Several to many > 10 to ≤ 30	Few to several ≤ 10
<u>Severe</u>	<u>Severe</u>	Neumrous and/or extensive > 30	Many > 10

Clinical variants :

1) Tropical acne :

- In tropics. in black face 1st at
- Nodulocystic acne affecting chest-back & arms (sparing) to face.

Clinical Types of acne

Clinical variants of acne

- Acne variants**
- 1 Acne Conglobata
 - 2 Acne Fulminans
 - 3 Neonatal acne
 - 4 infantile acne
 - 5 Post-adolescent acne
 - 6 acne excoりée
 - 7 cosmetic / pomade acne
 - 8 acne Detergiens

- 9 acne mechanica
- 10 premenstrual acne
- 11 occupational acne (chlorance)
- 12 senile (solar) comedones
- 13 Drug-induced acne = acne medicamentosa
- 14 pyoderma faciale
- 15 solid acid oedema

Salicid

① acne Conglobata

Blackhead

Conglobata: Shaped in Rounded mass or ball.

Incidence

Mainly in young Males.

Site

- usually on
- 1 Trunk
 - 2 Face, cheeks
 - 3 limbs

C.P.

Sever form of inflammatory characterized by

nodulocystic acne

① nodules

② Tender large cysts that may fuse to form multiple draining sinuses / suppuration

③ Grouped multiple fused blackheads (double or triple)

④ scarring of keloid type is present

⑤ No systemic manifestations.

* Many patients don't have preceding A.V lesions.

for acne

Acne Conglobata

differentiated from acne vulgaris

by its occurrence later in life
unremitting course.

* Follicular occlusion triad

- ① acne conglobata
- ② Hidradenitis suppurativa
- ③ Dissecting cellulitis of the scalp
- ④ pilonidal cysts.

acne conglobata
abscess, grain
in mat, axilla in female

Primary defect

- ① alteration of keratinization within the sebaceous follicle
- ② leakage of Retained sweat into tissue surrounding plugged eccrine ducts.

nodulocystic
acne

Treatment

- Start oral isotretinoids immediately
- not require oral steroids ~~XXXX~~

PAPA Syndrome

Syndrome

autosomal dominant autoinflammatory disorder.

① Sterile
Pyogenic
arthritis

Pyoderma
gangrenosum

acne conglobata

PPA

Prized skin
ulcers

Acne fulminans
Pyoderma Gangrenosum
Steroids

② Acne Fulminans

= acute ^① febrile ^② ulcerating ^③ acne conglobata
with ^④ polyarthralgia and leukemoid reaction ^⑤
= acne maligna

• Most severe form of acne

Incidence
Sex
Age

• Very Rare
• almost exclusively in young Men

only → jipij

C.P

① Sudden onset of severe nodulocystic acne, suppurative acne lesions + systemic manifestations
• Patients usually have mild to moderate acne prior to onset of acne fulminans

ulcers
in

• Initially it resembles acne conglobata with presence of highly inflammatory lesions
• Sites → on upper chest, back, with variable involvement of face

Characteristic morphological features

Formation of haemorrhagic

Nodules, Plaques

→ that undergo suppurative degeneration

leaving ragged ulceration

base of ulcer is filled with

gelatinous
neutrophilic debris

It
Ulcerulitis

- ① acute conjunctivitis
- ② acute blepharitis
- ③ G- α deficiency
- ④ pyoderma gangrenosum

various systemic manifestations

- ① fever
- ② arthralgia
- ③ Malaise, fatigue
- ④ myalgias
- ⑤ hepatosplenomegaly
- ⑥ osteolytic bone lesions



Lab. findings

- ① Anemia
- ② Leucocytosis
- ③ ↑ ESR
- ④ proteinuria

Treatment

- ① Bed rest, hospitalization
- ② Surgical debridement, warm compresses of 20-40°C saline solution
- ③ Topical antibiotic and cleanser

↳ medicated soap

Topical high-potency corticosteroids in the active phase of the disease or interlesional cort.

- ④ Systemic P → isotretinoin alone or Combined with systemic corticosteroids

- ⑤ NSAIDs for myalgias

- ⑥ Immunosuppressant (azathioprine)

- ⑦ Dapsone

- ⑧ Systemic antibiotics (limited efficacy)

SAPHO Syndrome

- S: Synovitis
 - A: acne
 - P: Pustulosis
 - H: hyperostosis
 - O: osteomyelitis
- Bone

(acne fulminans, acne conglobata, Pustular psoriasis, palmoplantar pustulosis)

Compare

PPPP

	acne fulminans	acne conglobata
1 sex	exclusively Men	Men & women
2 age	13-16y	20-25y
3 onset	Sudden acute febrile ulcerating	Slow
4 site	Face, Neck, Chest, back	
5 comp	haemorrhagic ulcers, crusting, suppurative → Ragged ulcers	Nodules, cysts, polymorphous comedones → sinuses, tract
6 systemic manifestations	very common autoaggressive fever, malaise HSM myalgia	uncommon X
7 Response to therapy	No (limited) to AB ↓ isolation ± CST Topical potent CST antibacterial cleanser, medicated soap hospitalization	Yes ✓ isotret No CST (usually not used CST) XX
		PAPA

③ Pyoderma faciale (Rosacea fulminans)

• no previous history of acne

Sex
F.P

• Post adolescent girls (20-40y)

Reddish

Cyanotic

erythema

with abscesses, cysts

Distinguish from acne

by ① absence of comedones. XX

② Rapid onset

③ fulminant course

④ absence of acne on back, chest X

Rapid

oral steroids

followed by isotretinoin

Preadolescent Acne

- Neonatal
- Infantile
- childhood / Juvenile

④ Neonatal acne

(neonatal cephalic pustulosis)

Malassezia

incidence

up to 20% of newborns

may be present at birth or develop during the first few months of life

Sex

more common

in males

Pathogenesis

Related to hormonal activity in utero

An inflammatory response to Malassezia species (Furfur)

have been proposed as the etiology.

disorder

Renowned as

neonatal cephalic pustulosis

additional support for

this view → clinical response to Ac with Topical 2% ketoconazole cream

A9A9

Cause It is relatively mild, Regress spontaneously in most cases by age of 6 months
 not associated with significant scarring or ↑ incidence of acne in later life
 X X X

- C-P**
- Facial papules or pustules
 - Typically No Comedones X X
 - Sites** • primarily on cheeks, Nasal bridge
 (but forehead, chin, neck, upper trunk can be also involved)

- R**
- No Treatment except Reassurance for parents and normal activity
 - Topical imidazoles (Ketoconazole 2% cream)
 (Nizoral cream)

after birth neonatal seb-
 excretion rates
 tends to match
 high levels seen in
 their mothers

Infantile acne

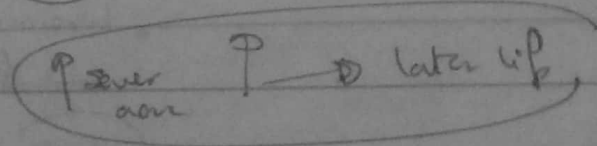
age usually begins bet 3rd, sixth month of life (persist beyond 4 weeks or have an onset later)
Sex 3-6m

- more common in males
- It is Rare

- C-P**
- Can be severe with nodules, cysts
 - In contrast to neonatal acne → Comedo is prominent
 (pitted) Scar can develop
 - Some cases associated with Virilizing Tumors

and neonatal

Cause • Resolves within 1-2 years, Remains quiescent until around puberty
 or may persist into adulthood



Pathogenesis hormonal imbalances intrinsic to this stage of development (elevated LH, Testosterone, DHEA)
 These androgen levels normally ↓ by 6-12m of age
 maternal hormones play minor role

Re

- Topical Retinoids
- Benzoyl peroxide

First line of Rx

oral antibiotics → erythromycin, azithromycin
 (helpful in more severe inflammatory condition)

Postadolescent acne

age: inflammatory acne persisting beyond 25 years of age

Sex: more common in females

C.P: Tender, deep seated Papulonodules with premenstrual flares/good response to hormonal Rx

Sites:

- lower $\frac{1}{3}$ of face
- Jawline
- Neck

$\frac{1}{3}$ of affected females → have other signs of hyperandrogenism

- female pattern baldness
- Insulin Resistance
- Acanthosis Nigricans
- obesity
- hirsutism
- ovarian infarctes

check → LH
 → FSH
 → DHEA-S
 → Free testosterone

Total Testosterone

Neonatal acne	Infantile acne (3-6m)
<p>May be present at birth or develop during the 1st few months 1st few months</p> <p>More common in males</p> <p>Mild & regress spontaneously by the age of 6 months</p> <p>Not associated with significant scarring or increased incidence of acne later in life</p>	<p>Begins between 3rd & 6th month</p> <p>More common in males</p> <p>Severe nodules & cysts & may persist to age of 5 years Persist or resolve within 1-2 y</p> <p>Associated with significant scarring or increased incidence of acne later in life</p> <p>Associated with <u>villilizing tumors</u></p>
<p><u>Pathogenesis:</u> (neonatal cephalic pustulosis)</p> <p>Species of malassezia</p> <p>Response to topical ketoconazole</p> <p>Sebum excretion: high level</p>	<p>Intrinsic hormonal imbalance</p> <p>↑ testosterone, LH, DHEA</p> <p>maternal hormones → minor role</p>
<p>① papules, pustules → cheeks, nose, bridge</p> <p>3/4 No scars, No ↑ acne later in life</p> <p>7/8 No comedones</p>	<p>• severe nodules, cysts</p> <p>3/4 comedones</p> <p>• pitted scars</p> <p>• ↑ incidence of acne later in life</p>
<p>② Retinoids</p> <p>• Topical ketoconazole 2%</p>	<p>• Topical Retinoids, Benzoyl peroxide</p> <p>• systemic antibiotics</p>

Compendium Acne, non-infectious

Age group	Location	Morphology	Sex
• Neonates	Nose, cheeks, forehead	✓ No comedones (Papules or pustules)	more in <u>males</u>
• Infants	Face	✓ No scarring. ✓ prominent comedones • inflammatory • no nodules, cysts ✓ Pitted scar	Common in <u>males</u>
• Preteens	Centrifugal	Comedonal	Both
• Teens	Face, Trunk	Mixed	Both
• Adults	<u>perioral</u> , <u>jawline</u> , <u>chin</u>	<u>inflammatory</u>	<u>women</u>

Premenstrual acne

- Papulo-pustular lesions weeks prior
- estrogen dominant contraceptives → ↓ acne
to gestation
pregnancy & post

excoriated acne (Acne excoriée des jeunes filles)
Pick (Pickier's acne)

* girls who have suppurative typical mild acne.

- * C.P.
 - Associated with excoriated lesions
 - Primary acne is small or even nonexistent but the patients have a compulsive habit of picking, squeezing them.
 - scratching, erosions, crusting
 - atrophy, scarring

• It is subset of neurotic excoriations associated with

OCD
anxiety disorder
personality disorder

Q

① Treatment of acne

② use of antidepressants or psychotherapy
(doxepin, SSRIs)

→ Obsessive Compulsive

Acne mechanica

Physical Trauma

• Secondary to Repeated Mechanical, frictional obstruction of pilosebaceous outlet.
• acne at sites of

Physical Trauma → Tight brasseries, Straps
→ Rubbing by helmets, chin straps
→ collars

on Neck of violin players
(Fiddler's Neck)

Repetitive Trauma from violin placement on lateral side of Neck → well defined lichenified
→ hyperpigmented plaques
→ interspersed with comedones
linear geometrically distributed areas
Suggest acne Mechanica.